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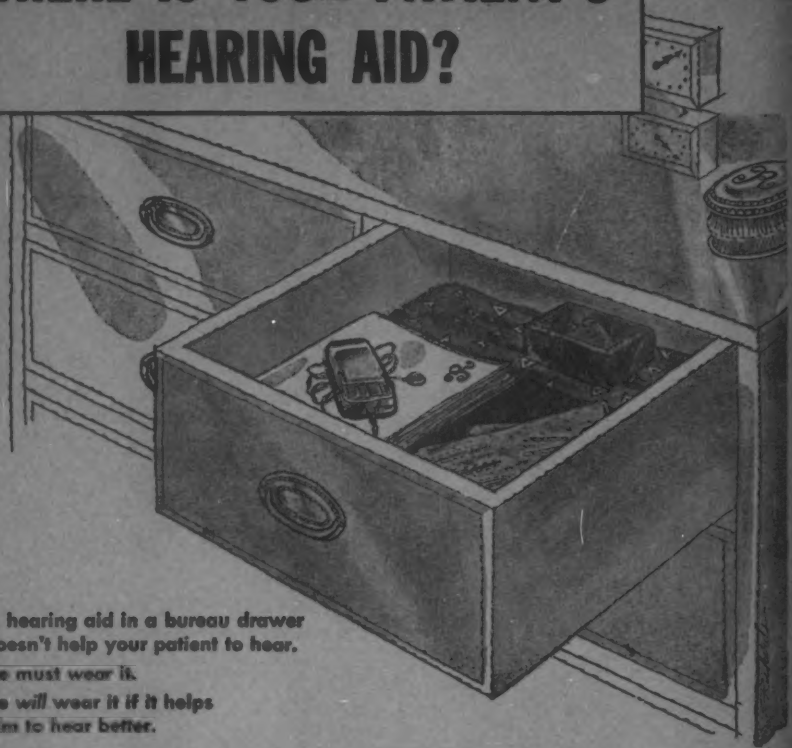

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VOL. LXIV AUGUST, 1954. No. 8

MORTALITIES AND MORBIDITIES FOLLOWING 20,000 TONSIL AND ADENOIDECTOMIES.*

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This is a discussion of the mortalities and morbidities following approximately 20,000 tonsil and adenoidectomies performed in a 30-year period by general practitioners and otolaryngologists, in Portland, Maine, a community of some 100,000 persons. Such a discussion is important, because these operations are considered by both the medical profession and the general public to be entirely "safe" and without mortality or morbidity; furthermore, this study is important since in the United States most tonsil and adenoidectomies are performed in communities of this size or smaller.

There is no criticism intended or implied, of the physicians who cared for the cases described in this article, and there is no question that the state of their practice measured well up to that of the community at the time of the occurrences here reported.

Obviously, patients expecting to undergo this operation, as a minimum, should have a well taken history, physical examination, urinalysis and determination of the hemoglobin. Should there be any question of their physical condition, they should also be examined by a pediatrician or internist. Adults should have a chest survey X-ray.

An inquiry into the bleeding habits should be made in each case. This may be confirmed by noting healed wounds and scratches and one or more empty tooth sockets. Determination of the bleeding and coagulation time and platelet counts

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should be reserved for those cases toward which history points the finger of suspicion. Negative examinations are not always dependable.

Mrs. T., now aged 52, had a tonsillectomy 25 years ago. She had a definite history of excessive bleeding after tooth extraction on three different occasions. At three different examinations prior to her tonsillectomy her blood and platelet counts were within normal limits, as was her coagulation and bleeding time. For the three weeks subsequent to her tonsillectomy I spent most of my time stopping bleeding from her tonsillar fossae. As years went by she was hospitalized a number of times, subsequent to dental extractions, for bleeding which demanded transfusions. Sometime later she had a hysterectomy because of excessive flowing at her periods, and eventually had her spleen removed. Other than anemia following her bleeding spells, her blood showed no abnormality.

At the beginning of this 30-year period two children operated on by two different general practitioners who did no other surgery than an occasional tonsil and adenoidectomy, circumcision, or opening an abscess, died of hemorrhage that was not controlled at the time of operation. A sponge was sewed into the tonsillar fossa in one and a tonsil hemostatic clamp that made pressure in the tonsillar fossa and on the outside of the neck was used in the other. These patients were operated on in the sitting position under general anesthesia. No suction machines were available in the Portland hospitals at this time.

This raises the question: "Who should do tonsil and adenoidectomies?" Those physicians who have passed the Boards in Otolaryngology should be qualified; otherwise it is up to the conscience of the individual physician and the judgment of the staff or board of the hospital at which he operates. It would seem that at the present time all interns on a rotating service have the opportunity to learn the technique and the problems involved in the operation of tonsil and adenoidectomy.

It should be impressed again and again on all physicians learning to do tonsil and adenoidectomies that because the chance for mortalities and morbidities to occur is small, their responsibilities for meticulous care for the safety of the patient is greater. Most patients undergoing this operation are small children. Within the last 30 years the average has

dropped from eight to ten years to three or four, and these children, whether service or private, are some parents' precious offspring.

Tonsil and adenoidectomies are one of the few surgical procedures in which the wounds are universally left wide open in an infected field. The control of hemorrhage, immediate or secondary, is surgical. It cannot be accomplished by the application of hemostatic sera, solutions of silver nitrate or persulfate of iron. The bleeding point must be found and tied about a hemostat with the fingers, with a slip knot or with a suture, and the bleeding stopped.

Postoperative bleeding after a tonsil and adenoidectomy is possible up to the eighth or ninth day. It happens most frequently in the first 24 hours. Watchful care in this period is the duty of the nursing staff who should contact the surgeon immediately when postoperative bleeding occurs or is suspected. Fresh blood from the nose or mouth and vomiting of blood and blood clots are obvious. The patient's color, the quality and rate of the pulse and respiration should be observed at hourly intervals at least for the first 12 hours.

If any post-tonsil and adenoidectomy bleeding occurs, get the patient's hemoglobin at once; if it is below 60 per cent give the patient an appropriate amount of blood.

The surgeon should seek the origin of the bleeding. There is almost always a clot in the fossa involved, or in the adenoid region, but bleeding may come from a severed uvula or from a nose bleed. Occasionally bleeding may be stopped by removal of the clot and making pressure with a sponge wet with 1-1000 adrenalin, but it is usually better to reanesthetize the patient in the operating room, clean out the clots, and find and tie the bleeding point. Bleeding may come from a partially detached bit of adenoid which should be removed. Retraction of the palate may reveal the bleeding point. It may be safer to insert a post-nasal pack.

I have had two instances in which the blade of a LaForce adenotome passed beyond the basket and caused bleeding from the back of the septum.

The following case history shows the necessity of having the hemoglobin estimated after severe postoperative bleeding:

A boy, age six, was operated on by a skillful general practitioner and surgeon. Throughout the day he had bouts of postoperative bleeding. I saw him in consultation at about 8 p.m., and thoroughly examined his tonsillar fossae and adenoid region while he was under a general anesthesia; after he had vomited up one of those terrifying placentas of blood clots, there was no bleeding. His general condition was good, although he was pale. He died later that night from surgical shock. I believe that he would have been living now if his hemoglobin had been taken, and he was given an adequate transfusion.

The case history cited below illustrates the need for competent postoperative nursing care to detect concealed bleeding:

A child, age six, was operated on by a competent otolaryngologist. She was returned to the ward in excellent condition, and had no evidence of bleeding when visited in the afternoon. During the night she vomited a large amount of blood and died. An autopsy showed her intestinal tract filled with clots.

Sudden death following tonsil and adenoidectomy was formerly considered to be due to status thymo-lymphaticus. The thymus was presumed to swell and obstruct the airways. Studies by Mosher and others have proved that this cannot occur.

Death in the following two patients could have been due to a blood clot from the adenoid region occluding the glottis which might have slid into the pharynx before autospy. It is probable, however, that death occurred from cardiac arrest or ventricular fibrillation. These cases suggest that following a tonsil and adenoidectomy, the patient be kept in the operating room until he has reacted from his anesthetic and then be accompanied to the ward or room by an anesthetist, or preferably taken to a recovery ward equipped with an anesthetist's Laryngoscope, suction machine, and having an attendant trained in anesthesia.

A young male adult, was operated on by a careful general practitioner and surgeon. The patient left the operating room in excellent condition and was being taken up six floors to the ward. Either on the elevator or as he was being taken to the ward, he expired.

A six or seven-year-old child was operated on by a skillful otolaryngologist. The patient left the operating room in excellent condition although the anesthesia had been stormy, and was moved to a ward on the next floor. Just as he was being placed in bed he expired.

A two-year-old child had cardiac arrest just as the tonsil and adenoidectomy was completed. He was operated on by a competent otolaryngol-

ogist. Anesthesia had been induced by Vinethane followed by drop ether. The course of the anesthesia was not smooth. Two competent general surgeons immediately opened the chest and massaged the heart. The child lived twelve hours.

Hypoxia of even a mild nature may lead to increased myocardial irritability with the possibility of ventricular fibrillation, cardiac standstill and death. The preoperative use of atropine may protect the heart from vagal stimulation.

Should cardiac arrest occur, the left pleural cavity should be entered by a long incision, in the fifth intercostal space, the costal cartilages above and below may be cut, the hand introduced and the heart compressed at the rate of the operator's pulse.

Two deaths from anesthesia occurred in children at a small hospital primarily devoted to orthopedic cases but in which occasional tonsil and adenoidectomies were performed. The nurse anesthetist was capable of giving adequate ether anesthesia for such cases providing the surgeon kept careful check on the depth of the anesthesia. She was accustomed to sitting at the head of the table, having the operator on the patient's right, and have him control the tongue with a tongue depressor. Motor driven ether vapor was rarely used in this hospital. The otolaryngologist had just completed his training at one of the larger centers where many tonsil and adenoidectomies were performed and where the anesthetists were entirely accustomed to the demands of anesthesia for this procedure. He used a Davis mouth gag with a fixed tongue depressor and sat at the head of the table.

Anesthetists tend to forget that the best and safest anesthesia for tonsil and adenoidectomy in children is well given drop ether. I believe it would be well if every otolaryngological resident spent at least three months in the Department of Anesthesia, particularly, giving tonsil and adenoidectomy anesthesia. The older otolaryngologists watched the depth of anesthesia as carefully as did the anesthetists, for the responsibility for the patient's life was and is theirs.

Preoperative sedation in children with depressant drugs in sufficient doses to insure their arrival in the operating room in tranquil state may cause prolonged postoperative sleep or

depression. Gas oxygen or Vinethane gives safe and not too unpleasant induction to drop ether.

Intratracheal anesthesia may be used for adult tonsillectomies done under general anesthesia, if its advantage outweighs the possibility of laryngeal damage. It does not prevent access of blood and mucous to the tracheo-bronchial tree unless the intratracheal tube has an inflatable cuff which increases traumatic risk.

A junior otolaryngologist felt that intratracheal anesthesia would be ideal to use in children when teaching tonsil and adenoidectomy to an intern. The head of the Department of Anesthesia intubated one child and everything went well. The following week a nurse anesthetist had difficulty in tubing a child. A tracheotomy was necessary in the afternoon. Having performed some 70 or 80 tracheotomies for various causes, I have a great respect for the ability of a child's larynx to become edematous and cause an urgent dyspnea.

I have performed tonsil and adenoidectomies with gas oxygen alone, with cyclopropane, and with sodium pentothal, having first anesthetized the larynx, but I believe that drop ether or drop ether supplemented by ether vapor driven by a foot bellows is the safest.

In local anesthesia a death occasionally occurs from accidental substitution of a solution of cocaine for novocaine. Cocaine solutions should be colored, and be in charge of the Department of Anesthesia in the operating room. Novocaine with adrenalin in ampules is almost foolproof, but there are exceptions.

Mrs. X., age 32, was scheduled for a tonsillectomy under local anesthesia. The attending nurse had just come into my employ and had never worked at the hospital in which the operation was to be performed. I noted that the ampule was different than usual and received three affirmative answers that it was 1 per cent novocaine with adrenalin. After I had injected it, I was told that it was Sodium Diodrast. It made a good anesthesia and the recovery was uneventful.

Post-tonsillectomy lung abscesses were formerly considered to occur about once in five or six thousand cases. Seven have occurred in this area, all in the first half of the 30-year period. Six followed general and one a local tonsillectomy; they all occurred in young adults. One underwent external

surgery, the others cleared up with bronchoscopic drainage. Blood and mucous has been found in the trachea after both local and general tonsillectomies.

Palpable fremitus from mucous can be noted in the chests of many children when under anesthesia. One school of thought believes that lung abscesses come from infected emboli given off from the tonsillar fossae whose shapes continually change with the muscular action of the pharynx. Another feels that they are caused by inspiration of infected material when conditions are right. *A head low position with adequate use of suction minimizes aspiration.*

Foreign bodies occasionally lodge in the tracheobronchial tree during a tonsil and adenoidectomy. Prior to a general anesthesia the mouth should be searched for not only artificial dentures but also gum and other foreign substances. Loose teeth should be noted. The operator also should inspect his instruments for small loose parts. X-rays taken some two months after tonsil and adenoidectomies revealed teeth in the tracheo-bronchial trees of two children, both of whom were operated on by competent otolaryngologists. In neither of these cases was the loss of a decayed crown or of a deciduous incisor noted by the operator or anesthetist immediately after the operation. They could have been aspirated at a later date, but the presumption is that they were aspirated at the time of the operation, which was done in the prone or dorsal position. This again suggests the advantage of the low head.

A piece of rubber tubing covering the dental surface of a side mouth gag was aspirated. This type of gag is rarely used today.

Pieces of tonsil and adenoids have been aspirated. The use of a LaForce type of adenotome, frequent aspiration, and unremitting care should make such accidents unusual.

I have not adequate check on the number of cases of postoperative bleeding, bronchitis, pneumonia, infectious disease, acute otitis media, pyelitis, nephritis, and rheumatic flare-ups that have followed these operations. There have not been any deep abscesses following local tonsillectomy. One patient had a broken hamular process of his palate, another operated

on by a general practitioner received an injury to the lingual nerve when stopping bleeding which caused a loss of taste and sensation on one-half of the tongue, much to the patient's distress.

Asthma and allergy will not be cured by the removal of tonsils and adenoids. Asthma *per se* is not a contra-indication to ether anesthesia as ether is a bronchial dilator.

A boy, age six, who had severe asthmatic attacks, also had tonsils that all but met in the midline and marked difficulty breathing through his nose. I felt that the child would be safer in his asthmatic attacks with his tonsils and adenoids out. He was operated on, and it was my intention to keep him in the hospital for a week, but after four days with absolutely no asthma, his mother insisted upon taking him home to a neighboring town. Two days later he had a severe asthmatic attack and died. I do not feel that the operation had anything to do with his death.

One mild case of poliomyelitis with a quickly clearing paralysis in a leg occurred three weeks after a tonsil and adenoidectomy, which was performed in May, when there had been no other cases reported in this part of the State. Another patient developed poliomyelitis three weeks after a scheduled tonsil and adenoidectomy, which was not done.

SUMMARY AND RECOMMENDATION.

Nine deaths, four from hemorrhage, two at the time of operation and two from postoperative bleeding, three cardiac arrests, two anesthetic deaths, seven lung abscesses, three foreign bodies and a number of minor morbidities followed 20,000 tonsil and adenoidectomies performed by general practitioners and otolaryngologists in a 20-year period in a community of 100,000 people.

It is recommended: that interns on rotating services be taught to perform tonsil and adenoidectomies; that residents in otolaryngology spend at least three months in the Department of Anesthesia; that drop ether is the safest anesthetic for tonsil and adenoidectomy; that solutions of cocaine in the operating room be in charge of the Department of Anesthesia; that atropine be given preoperatively; that all patients having postoperative bleeding have a hemoglobin determination and have an adequate transfusion if their hemoglobin is below 60 per cent; that patients be operated on in a head low position; that the patient be kept in the operating room until he

has reacted from his anesthesia and be accompanied to the ward, room, or recovery ward by an anesthetist; that close postoperative watch be kept by the nursing staff for bleeding or vomiting of blood, and that the pulse, respiration, and color be noted at hourly intervals for the first twelve hours; that intratracheal anesthesia be limited to adults; that no preoperative sedation with depressant drugs be used in children; that if cardiac arrest occurs, the chest be opened and the heart massaged; that allergy *per se* is not a contra-indication to tonsil and adenoidectomy; and that the possible relationship of poliomyelitis to tonsil and adenoidectomy be further studied.

FIFTY-SECOND CONGRESS FRANCAIS d'OTO-RHINO-LARYNGOLOGIE.

The 52nd Congrèss Francais d'Oto-rhino-laryngologie will meet October 18 to 22 in the Grand Amphithéâtre of the Faculté of Médecine, Paris, under the presidency of Professeur J. Terracol, of Paris, and honorary president Professeur Lèon Binet, member of the Institut, Doyen of the Faculté of Médecine of Paris. A number of interesting papers will be given, among them, "La Thérapeutique par les ultra sons en Oto-rhino-laryngologie," by Professeur Portmann, with MM. Michel Portmann and Louis Barbe, as collaborators; "De la greffe cutanée libre en chirurgie otologique," by MM. Ombrédanne, Clerc, and Poncet.

AN EXPERIMENTAL STUDY OF THE SMALL BLOOD VESSELS OF THE SPIRAL LIGAMENT AND STRIA VASCULARIS OF LIVING GUINEA PIGS DURING ANAPHYLAXIS.*†‡

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INTRODUCTION.

The purpose of this paper is to describe a series of microscopic observations made upon the walls and intravascular cellular elements of the arterioles, capillaries, and venules of the spiral ligament and stria vascularis of living, anesthetized guinea pigs during anaphylaxis. No one has previously reported such a series of observations.

Several investigators, however, have studied small blood vessels in other tissues of living animals undergoing anaphylaxis. Insertion of transparent chambers into the auricles of the ears of rabbits enabled Abell and Schenck[1] to study arterioles, capillaries, and venules. During anaphylaxis contraction of arterioles, adherence of leucocytes to vessels' walls, emigration of leucocytes through capillary and venular walls, and blocking of capillary blood flow by white blood cell emboli were observed.

In a similar series of experiments Ebert and Wissler[2] noted sticking of leucocytes to arteriolar endothelium, swelling of arteriolar endothelium, localized constrictions and dilatations of arterioles, focal swelling and proliferation of venular endo-

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† From the Winthrop Foundation of the Massachusetts Eye and Ear Infirmary and the Allergy Laboratory of the Massachusetts General Hospital, Boston, Massachusetts.

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thelium, venular dilatation, red cell aggregates, platelet and white blood cell emboli and thrombi.

Thromboses were seen forming in retinal veins by DeMuro and Focosi[3] when they inoculated the vitreus of sensitized rabbits with the specific antigen.

Recently Burrage and Irwin[4,5] have reported changes in the small blood vessels of the livers and lungs of living mammals during the anaphylactic reaction. Contraction of the outlet sphincters of liver sinusoids resulted in storage of blood in the liver. No functioning hepatic arterioles were observed, and linear blood flow stopped in central venules.

Contraction of both pulmonary arterioles and venules was noted when death quickly followed the intravenous injection of the specific antigen. When death did not occur until five minutes or more after the shocking dose of antigen, dilatation pulmonary venules, contraction of arterioles, no open capillaries, aggregates of erythrocytes, formation of emboli and thrombi and leakage of red blood cells from small venules and capillaries were observed.

These experiments as a group at least indicate that changes in small blood vessels do occur when animals undergo anaphylaxis. These small vessels, furthermore, have an important role to play in the physiology of each tissue studied. Changes in them may well be of paramount importance.

MATERIALS AND METHODS.

Over 200 healthy guinea pigs weighing 200 to 1,000 grams were used in these experiments. Observations were made in 57 of these animals in which the blood vessels appeared normal and intravascular cellular elements formed no emboli or red cell aggregates. Each animal was sensitized by two 0.5 ml. intraperitoneal injections of 1-10 solution of crude, sterile egg white five days apart. No animal was presented with a shocking dose until 21 days or more after the initial dose.

In a sensitized and anesthetized animal the spiral ligament and stria vascularis were exposed according to the method described by Weille and associates.[6] The anesthesia (sodium pentobarbital 45 mg. per kg, of body weight) was a principal

cause of failure since the amount required for adequate narcosis was very close to the lethal dose; furthermore, subsequent doses were needed to maintain the required level of narcosis. In both instances, normal physiological relationships were altered to some extent. Unavoidable surgical trauma during exposure of the cochlea also led to failure. Careful and immediate hemostasis controlled excessive bleeding.

The next step consisted of the microscopic exposure of the spiral ligament. Light was provided by a 1,000-watt projection bulb and was transmitted to the cochlea by a fused quartz rod. This rod had a hollow tip through which Mammalian Ringer's solution at 38° C. flowed gently on the cochlea. To visualize the cochlea, a Leitz stereoscopic microscope with magnifications 48x was used. Attention was then placed on an area limited by the two bone partitions that separated a cochlear turn.

In these experiments, the third or fourth turn was worked on. During the fenestration the spiral ligament was easily damaged. To avoid trauma, the method was modified in the following manner: Using an electric drill with a polishing burr No. 5, the operator circumscribed an area of bone measuring 1 mm. in the horizontal plane and 0.5 mm. in the vertical plane of the cochlear base. The bone was thinned down to its endosteum. Then the circumscribed area plus the underlying endosteum were lifted up gently and removed with a dental scaler. Bone chips were thus avoided and clean sharp edges of the fenestra were obtained.

In order to administer the shocking dose to the animal, a No. 22 cannula was inserted into the external jugular vein and connected by means of a fine polyethylene tube to a syringe. Observations were made with a Leitz monocular, monobjective microscope provided with a 15x ocular and dry 11x objective. At the time of the observations, fogging of the surface lens of the objective was prevented by soft constant suction at the edges of the bulla cell. Such suction also removed any excess of Ringer's solution. Cinematographic records were taken at 8 frames per second when type A Ko-

dachrome was used, and 24 frames per second when Super X was used. As soon as the shocking dose was given, the animal began to convulse. This required frequent refocusing of the optical system.

OBSERVATIONS.

Vessels studied during these experiments included: the arterioles, the arteriovenous anastomoses, the capillaries, and the venules of the spiral ligament, as well as the capillary network of the stria vascularis. To be considered an arteriole of the spiral ligament a blood vessel had to fulfill certain qualifications. The vessel had to be in the spiral ligament, and it had to take the shape of a cone with the direction of linear blood flow going from the larger end of the cone toward the smaller. In general no part of any arteriole had a diameter greater than 60 microns.

The capillaries appeared in the spiral ligament as an intricate network of dividing and anastomosing cylinders receiving blood from arterioles. The arteriovenous anastomoses were vessels in the spiral ligament. They started off as branches of an arteriole and emptied directly into a venule with no intervening capillary network. At no point did such anastomoses look like cylinders. In the area of pigment cells were found the capillaries of the stria vascularis. These capillaries also formed a network of branching and anastomosing cylinders, and they too emptied into the venules of the spiral ligament. These venules took the form of cones with the direction of linear blood flow from the part of the vessel with the smaller diameter toward that with the larger diameter. Some of these venules ran horizontal to the field, and these in turn drained into venules perpendicular to the field.

During anaphylactic shock certain changes both in the walls of these vessels as well as in the intravascular cellular elements were noted. These changes were by no means consistent, but were more similar when they were compared in animals with the same degree of anaphylaxis. The degrees of anaphylaxis can be divided into three groups; a few animals died within two minutes after receiving the shocking antigen; however, most did not die until five or ten minutes

and a few recovered. Obviously there is considerable overlap, and these groups are not clear-cut.

Changes in the calibers of vessels were marked when a sensitized guinea pig died quickly after the shocking dose of antigen. Arterioles contracted. Indeed contraction often was so marked that arterioles were not observed. Figure 1 shows a field with an arteriole of the spiral ligament before anaphyl-

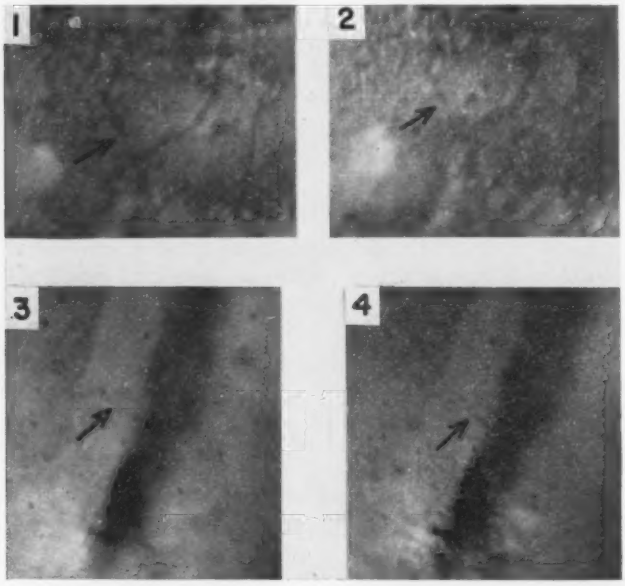

Plate One. Fig. 1—Arrow Points to arteriole before anaphylaxis. Fig. 2—Arrow points to same arteriole as in Fig. 1 after anaphylaxis. Fig. 3—Arrow points to a venule before anaphylaxis. Fig. 4—Arrow points to same venule as in Fig. 3 after anaphylaxis.

axis, and Fig. 2 shows the same field during anaphylaxis. Similar behavior of the arteriolar part of arteriovenous anastomoses was observed. Venules contracted, but in only two experiments did they disappear from view. Capillaries of the spiral ligament and those of the stria vascularis were never well visualized because of limitation of magnification. When either set of capillaries were observed both before and during

anaphylaxis, the capillaries tended to disappear. Since the walls of capillaries were never well outlined, the apparent disappearance could be due to cessation of blood flow because of arteriolar contraction. Emboli and thrombi were not seen when the reaction progressed rapidly to death.

Usually the sensitized guinea pig did not die until five to ten minutes after the shocking dose of the specific antigen. Again the arterioles contracted, but rarely did they contract tightly shut. The arteriolar parts of arteriovenous anastomoses also contracted. Venules could contract slightly, but the general pattern was dilatation. Figure 3 shows a field with venules before anaphylaxis, and Fig. 4 shows the same venules dilated during anaphylaxis.

The venular portions of the arteriovenous anastomoses followed the venular action. Capillaries of the spiral ligament and stria vascularis disappeared. At least they could not be visualized. Masses highly refractile to light appeared on both the arteriolar and venular sides of the circulation. Aggregates of erythrocytes were also seen. Often blockage of a venule or arteriole by one of these highly refractile masses or one of the red cell aggregates was observed. Experiment No. 1 illustrates many of the above points.

EXPERIMENT NO. 1

Male Guinea Pig—Weight 1,050 grams; egg white sensitive.

Date—August 25, 1953.

10:00 A.M.—42 mg. of sodium pentobarbital intraperitoneally.

10:30 A.M.—Surgery started.

2:30 P. M.—Observations started.

2:35 P. M.—Two branching arterioles; rapid linear blood flow. Two arteriovenous anastomoses; rapid linear blood flow. Three venules running horizontal to field; rapid linear blood flow. Several venules running vertical to field; rapid linear blood flow. Actively flowing capillaries of stria vascularis and spiral ligament.

2:40 P.M.—0.1 ml. egg white 1-10 intravenously.

2:41 P.M.—Contraction of arterioles and arteriolar parts of arteriovenous anastomoses; venules dilating.

2:44 P.M.—Irregular shaped emboli highly refractile to light observed in arterio-venous anastomoses and venules as well as aggregates of erythrocytes. These emboli stretched the arteriolar walls as they passed through, but the walls returned to constricted phase after emboli passed.

2:46 P.M.—Arterioles contracted; no linear blood flow.

2:47 P.M.—Emboli stuck in venules, blocking flow.

2:48 P.M.—Capillaries not visualized.

2:51 P.M.—No apparent linear blood flow in any vessel.

2:52 P.M.—Guinea pig died.

Anaphylaxis did not prove to be fatal in a few guinea pigs. The arterioles and arteriolar part of arteriovenous anastomoses constricted. Venules dilated. The capillaries either showed no change or disappeared from view. Emboli highly refractile to light were seen in all vessels, and aggregates of erythrocytes were found in all vessels. Both types of emboli appeared more evident on the venous side of the circulation. Frequently emboli would plug the lumen of small arterioles and venules. These emboli might, therefore, be classified as thrombi. If the animal lived, the circulation returned to normal. Emboli disappeared and arterioles and venules returned to their original calibers. In no instance, however, did the thrombi ever disappear. It must be pointed out that no field was visualized for more than four hours after the shocking dose. Experiment No. 2 will point out several of these factors.

EXPERIMENT NO. 2

Male Guinea Pig—Weight 975 grams; egg white sensitive. Date—August 26, 1953.

9:00 A.M.—33 mg. of sodium pentobarbital intraperitoneally.

9:30 A.M.—Surgery started.

12 noon—Observations started. Arteriole: rapid linear blood flow. Arteriovenous anastomoses; rapid linear blood

flow. Two venules running horizontal to field; rapid linear blood flow. One venule running vertical to field; rapid linear blood flow. Capillaries of stria vascularis; network.

12:15 P.M.—0.3 ml. egg white intraperitoneally.

12:16 P.M.—Constriction of arterioles and arteriolar part of arteriovenous anastomoses. Venules dilated. Capillaries: no change. Emboli highly refractile to light in all vessels. Aggregates of red cells in all vessels.

12:17 P.M.—Flow in all vessels. Emboli in all types of vessels under observation.

12:18 P.M.—Emboli have plugged one arteriole and one arteriovenous anastomsis (*i.e.* thrombi).

12:21 P.M.—Arterioles dilating. Flow in all vessels. Fewer emboli.

12:40 P.M.—Vessels as at start of experiment before shocking dose.

3:00 P.M.—No emboli. Thrombus still in one arteriole, and in one arteriovenous anastomosis.

DISCUSSION.

In any series of experiments certain experimental conditions are introduced which may well effect end results. It was necessary to anesthetize deeply each guinea pig with sodium pentobarbital. Unfortunately, with sodium pentobarbital the margin between adequate narcosis and death was narrow. Sodium pentobarbital does affect to some extent small blood vessels, but no better agent is available.

Operative procedures, used to expose the cochlea and to fenestrate one of its turns, were extensive. If the spiral ligament or its blood vessels were damaged, the guinea pig was discarded. Thus anesthesia and surgical trauma accounted for most of the failures. Once a successful fenestra was made, it was imperative to keep the exposed tissue moist and at normal temperature.

The amount of light needed for observation was large. This light itself was responsible for considerable heat. The problems of heat and moisture were controlled in part by a gentle

flow of Mammalian Ringer's which emitted from the very tip of the quartz rod at a constant rate and temperature. Controls to date are crude and much remains to be done in this phase.

The problem of magnification remains. Objectives of 20x to 90x have short working distances, and are so large in diameter that they can not be placed near the fenestra because of the narrowness of the bony vault about the cochlea. Until adequate means of magnification are available, accurate observations upon the capillaries must wait.

In spite of inherent difficulties over fifty successful preparations were studied. All observations were made on these animals. The blood vessels described under observations were found in each animal. The intravascular cellular elements formed no emboli or thrombi until anaphylaxis was induced.

Certain changes both in the calibers of the small blood vessels of the spiral ligament and in the intravascular cellular elements occurred consistently during anaphylaxis which is a general systemic reaction depending on antigens and antibodies. During anaphylaxis the main changes in calibers of vessels appeared to be constriction of the arterioles of the spiral ligament and dilatation of the venules.

At times the arterioles constricted tightly shut. Since these vessels quickly returned to normal size if the animal survived anaphylaxis, it is difficult to determine how important such constriction and dilatation might be. The formation of emboli and thrombi within the vessels appears to be of more importance. Even though most of the emboli disappeared once an animal survived, thrombi plugging certain of these vessels did not disappear. Such plugged vessels would be unable to perform their usual function. Surrounding tissue might consequently be damaged.

SUMMARY.

1. Anesthetized guinea pigs sensitive to egg white can die within 2 or 3 minutes after the shocking dose of antigen. The changes noted in the blood vessels of the spiral ligament included constriction of arterioles and initial constriction followed by dilatation of venules.

2. When death did not occur until several minutes after the shocking dose, beside constriction of arterioles and dilatation of venules of the spiral ligament, emboli and thrombi were observed in both arterioles and venules.

3. When the guinea pig recovered from anaphylaxis, thrombi in the vessels did not disappear.

4. The phenomena observed in the inner ear of the guinea pig during anaphylaxis may or may not apply to other mammals, but they are of interest since such observations in the labyrinth have not been previously reported.

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ANNOUNCEMENT.

An introductory course in Reconstructive Surgery of the Septum and External Nasal Pyramid will be given, under the direction of Dr. Maurice Cottle, March 19 through 26, 1955, at the University of Oregon Medical School. This will be sponsored by the Department of Otolaryngology.

THE SURGICAL TREATMENT OF THE ATRESIA AURIS CONGENITA; A CLINICAL AND HISTOLOGICAL REPORT.*†

By Professor L. RUEDI
Zurich, Switzerland.

With the aid of fenestration technique and the use of antibiotics, the prospects of surgical treatment of congenital atresia of the ear have become more favorable. Of course, the opinions concerning the indications to operate and the technique to be used are still not quite settled. M. Ombrédanne operates preferably on cases of one-sided atresia in order to relieve these mentally depressed patients and to obtain a reserve of hearing. He creates a new meatus, and in the same stage of the operation the horizontal semicircular canal is fenestrated. G. L. Pattee performs the operation primarily on patients who are bilaterally affected. After making a new external meatus the incus is removed. By this method the stapes should become mobile, and the hearing should improve as with a successful fenestration. R. F. Guilford and George E. Shambaugh refrain from removing the incus where there is only a slight alteration of the ossicles and the incudo-malleolar articulation seems functionally normal. The new meatus ends in a skin flap which seals off the tympanic cavity and covers the auditory ossicles. In bilaterally affected patients, DeGraaf Woodman recommends a two-stage operation as suggested by F. Altmann. In the first stage a new meatus is made, and the chain of auditory ossicles is disconnected. If after the first stage the hearing gain is insufficient, a fenestration of the horizontal canal will follow in a second operation.

Our experience with the operative treatment of congenital atresia of the ear, collected since 1948, concerns four female and nine male patients, ranging in age from 7 - 39 years

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(see Fig. 1). Twelve of these patients were afflicted bilaterally and all of them had intact inner ear function, thereby fulfilling the indication to operate. One patient with one-sided atresia had to be operated upon because a perception deafness gradually developed in the second previously normal ear. In six patients both ears and in seven patients, one ear, were operated. The best cosmetic results obtained by operation are in our opinion surpassed by auricle prosthesis of plastic material; therefore, this aspect of the atresia problem will not be treated in the current discussion.

Nr	Sex	Age	Atresia		Pneumatization		Further Malformation	Debilitas	Heredity	Ombrédanne		Pattee		Guilford		Altmann-Woodman		[illegible]		
			right	left	right	left				right	left	right	left	right	left	right	left	right	left	
1	♂	27	medium	medium	+ +	+ +		+				XII 48	I 49						II 54	
2	♂	39	medium	medium	+ +	+ +		+				XII 49								
3	♂	11	medium	mild	+	O		+	1 Atresia (Father Gen.)			VII 54	II 53							
4	♂	8	mild	mild	+	+		+							IV 52					
5	♀	17	mild	medium	+ +	+ +						VII 53	VIII 53					I 54		
6	♂	26	medium	medium	+	+ +			1 Brother Atresia			XI 49	XI 49							+
7	♂	10	medium	medium	+ +	+ +		+ Epilepsy				X 52								
8	♂	36	medium		+ +	+ +						I 52				XII 52				
9	♂	10	medium	medium	O	O	cleft palate	+				II 53								✝
10	♀	21	medium	medium	O	O	cleft palate Dysost. Acro-Facialis					II 50	II 50					II 54		
11	♂	7	severe	severe	O	O	cleft palate Dysost. Mand.-Facialis	+		II 54			VII 53							
12	♀	8	medium	medium	O	O	Choanal-Atresia Dysost. Mand.-Facialis	+				I 54								
13	♀	16	severe	severe	O	O	cleft palate Dysost. Mand.-Facialis	+	1 cleft palate (Father Gen.)			VI 53								

Fig. 1. Schedule.

As to the severity of malformation our cases can be subdivided, according to H. Marx and F. Altmann, by means of the ear status, roentgenological examination and operative findings, in the following manner:

Four ears show atresia of mild degree.

Seventeen ears show atresia of medium degree.

Four ears show atresia of severe degree.

The characteristic anatomical changes in these various degrees of malformation have been excellently described by F. Altmann.

In a first group of nine patients (above the dotted line) there exists in one case an additional malformation, a cleft

palate. In this group, roentgenological examination of the mastoid in eight of nine cases revealed good to very extensive pneumatization. This confirms the observation of F. Altmann that "in almost all cases with hypoplasia of the middle ear, alone or combined with hypoplasia of the tube, there was a mastoid process of normal size, and as a rule with good development of the pneumatic cells."

In the second group of four cases (below the dotted line), in addition to the congenital atresia there was malformations, mainly of the head and, in one case, also of the extremities. In these four cases the mastoid process is almost completely absent, and the pneumatic cells could not be seen either roentgenologically or during the operation. In all four cases of the second group the malformation syndrome known as mandibulo-facial dysostosis as described by Treacher-Collins and by A. Franceschetti and P. Zwahlen, was present.

The Franceschetti-Zwahlen syndrome differs from O. Crouzon's cranio-facial dysostosis in that the former has no malformation of the cranium. F. R. Nager observed two of our cases (Nos. 10 and 13) for many years. Both cases were extensively reported by F. R. Nager and J. P. de Reynier, in the monograph "The Hearing Organ in Congenital Malformation of the Head."

We will, therefore, restrict ourselves to a short description of the changes in the facial part of the skull which were manifest in our four cases, in addition to the atresia of the ear.

Case No. 10 concerns a girl born in 1929. At birth the child showed malformations of both auricles, abnormalities of both thumbs and a cleft palate. The cleft palate was operated upon when the child was two and four years of age. The child began to speak at the age of one and one-half years. Of normal intelligence, she successfully attended several schools. In addition to the congenital atresia, malformed auricles and absence of the meatus, the face (see Fig. 2) shows a slight hypoplasia of the maxilla and symmetrical slanted position of the eyes. The thumbs (see Fig. 3) are absent on both hands. The right arm is rigidly fixed at a right angle and can be supinated and pronated only in a limited manner. Roentgenologically the cranium is normally configurated, the sinuses are small, the zygomatic arches hypoplastic and the maxilla and mandibula are somewhat underdeveloped. In the roentgen picture of the upper extremities there is a synostosis of the radius and ulna in the proximal third. In both hands the thumb is absent and also missing are the phalangea, metacarpal I, multangulum majus and the naviculare. The multangulum majus is fused with the naviculare.

Fig. 2.

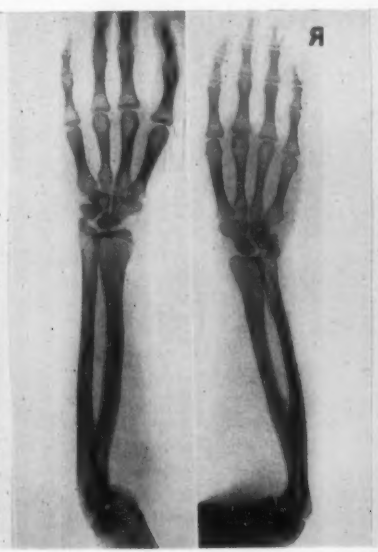

Fig. 3.

Fig. 4.

Fig. 2. Case No. 10. Female, 21 years, Atresia auris congenita with dysostosis akro-facialis.
Fig. 3. Case No. 10. Roentgen Picture of the upper extremities.
Fig. 4. Case No. 13. Female, 16 years, Atresia auris congenita with dysostosis mandibulo facialis.

To summarize there exists a congenital ear atresia, a mandibulo-facial dysostosis, combined with malformation of the extremities.

Case No. 11 concerns a boy born in 1945 with bilaterally malformed auricles, absent meatus, a cleft palate and malformation of the facial skull. The intellectually retarded child began to speak at four years of age. The cleft palate was operated upon in 1952. On the face there may be seen a hypoplasia of the upper and lower jaw and a slanted position of the eyes.

It is again a case of mandibulo-facial dysostosis (A. Franceschetti and P. Zwahlen).

Case No. 12 is a girl born in 1946 afflicted with a congenital atresia of the ear which is restricted bilaterally to the middle ear. The large, protruding auricles are normal. The outer ear canal is present on both sides. On both sides a pale red, translucent ear drum with severely reduced mobility can be seen. Hearing tests show a severe sound conduction deafness on both sides.

In addition there is a choanal atresia on the left side and an underdevelopment of the facial skull showing a mandibulo-facial dysostosis.

Case No. 13 is a girl born in 1937, who at the time of birth showed deformed auricles, absence of the outer ear canals, a cleft palate and a severe deformation of the facial skeleton. At the age of one and one-half years the child could barely speak. At the age of two and four years the cleft palate was operated upon. In Fig. 4 the typical "bird" face can be recognized with a prominent hooked nose, hypoplasia of the mandible, hypoplasia of zygomatic arches, slanted position of the eyes and coloboma of both lower lids. The roentgen picture of the skull shows an internal hydrocephalus with normal sutures compensatory enlargement of the posterior fossa and a steep position of the base of the skull.

Here again a very pronounced case of mandibulo-facial dysostosis is present.

In the four cases of our second group with malformations of the ear and facial skeleton the following abnormalities are found:

(a) a bilateral congenital atresia of the ear of medium to severe degree;

(b) a hypoplasia of the zygomatic arches and mandible;

(c) eye lids slanted down laterally with a peculiar bending of the lateral third of the lower lid and coloboma formation;

(d) cleft palate in three of four cases, in one case a choanal atresia;

(e) Case No. 10 showed a deformation of the upper extremities in addition.

A certain parallel can be drawn to acro-cephalo-syndactyly observed by E. Apert and to cephalo-syndactyly described by A. Vogt, who in 1930 called attention for the first time to the combined occurrence of a cranio-facial dysostosis; that is, Crouzon's disease, with symptoms of Apert's disease. In view of these combined malformations, F. R. Nager has proposed the term acro-facial dysostosis for our Case No. 10. Pathogenetically we must assume a disturbance of the organizer of the trunk as well as of the head.

The intellectual development was clearly retarded in nine of the 13 cases with ear malformation. In the group with uncomplicated atresia (possibly combined with a cleft palate), three out of nine patients are normally intelligent; in the groups of complicated ear and face malformations, only one out of four.

There is as yet very little known as to the cause of ear and skull malformation. F. Altmann has recently mentioned genetic and environmental factors that could damage the organizer of the head in the early embryological state. Two of our patients have blood relatives with congenital malformation of the ears. Cleft palates occurred in the families of three other patients, so that genetic factors would appear to play a role in these cases.

Finally — Fig. 1 gives information concerning the respective surgical therapy. Since we were followers of Pattee's method, 12 of the 19 ears were operated upon in that fashion. [In Case No. 4 only slight alterations of the tympanic cavity and a mobile malleus-incus articulation were found, which is the reason that the ossicles were left intact (modification, Guilford-Shambaugh).] In Case No. 11 with combined ear-face malformation the one-stage method of M. Ombrédanne was used. F. Altmann-De Graaf Woodman's two-stage operation was performed once and tried once, and finally in three late re-examinations, secondary fenestration was done.

Technically, operating on the uncomplicated atresia with good pneumatization of the mastoid presents no special diffi-

culties. The operation is very difficult, however, in cases of mandibulo-facial dysostosis. As a rule there is no mastoid process, the guiding pneumatic cells, the antrum and frequently also the aditus ad antrum are absent. The widely exposed dura of the middle fossa and the sino-dural angle serve as guiding points for locating the middle ear cavity, which is always very small. The middle ear cavity is found by following the exposed dura with the burr in a ventrally-medial direction. The reasons for certain modifications in our surgical procedure can be seen from the critical observation of our early and late results:

Ten ears operated with the Pattee method (see Fig. 5) (Case 4 with Guilford-Shambaugh modification), showed a good to sufficient hearing gain in the immediate post-operative

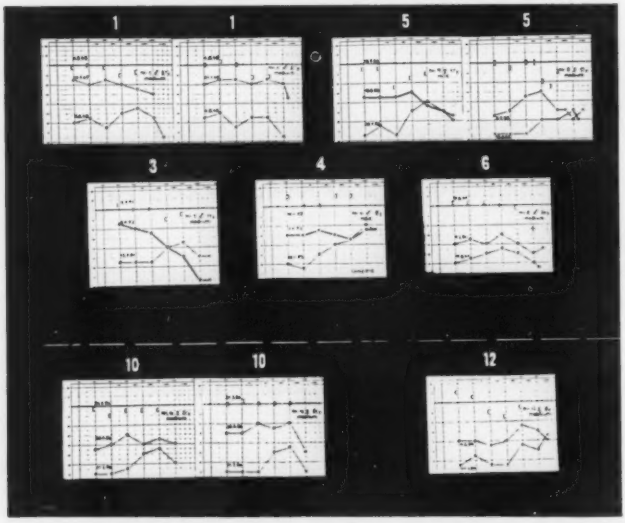

Fig. 5. Audiograms of atresia auris congenita showing good or sufficient hearing gain after the Pattee operation.

period. The audiograms of uncomplicated atresia are above the dotted line; the audiograms of the complicated cases with ear, face and skull malformations are below the white line.

Eight ears operated according to Pattee show insufficient or no hearing gain (see Fig. 6). There are eight failures, as compared to 10 early successes of the Pattee operation. The operative failures of cases Nos. 11 and 13, with combined face and skull malformation, can be explained by the extensive deformity in the ear region.

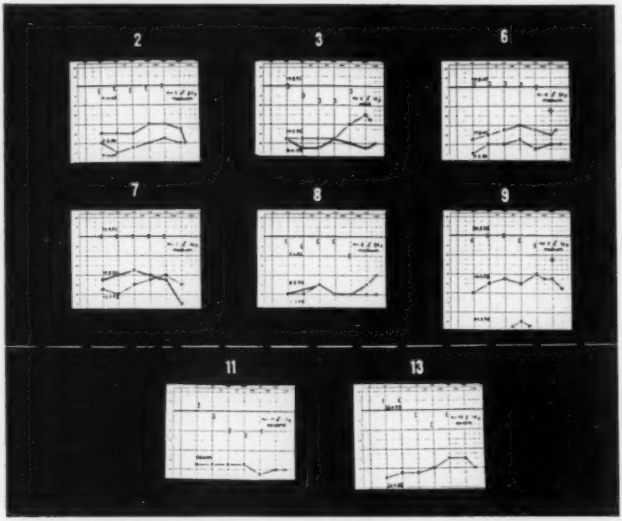

Fig. 6. Audiograms of atresia auris congenita showing insufficient hearing gain after the Pattee operation.

In Case No. 13 (right ear) after hours of searching, a small slit was found, filled with red granulation tissue and rudimentary ossicles. The stapes could not be found. From all appearances the tympanic cavity was present only as a small slit, so the operation was terminated without the formation of a plastic meatus.

In Case No. 11 (left ear) where the antrum and aditus were absent, a very small middle ear cavity was finally found and a rudimentary incus removed from a fold of mucous membrane. Subsequently the exposed middle ear cavity and the new meatus were lined with epithelium in the typical manner by a pedunculated skin flap.

The negative operative results in the uncomplicated atresia (above the dotted line) cannot be conclusively explained by the operative findings.

Two patients of this group (Case No. 6, left ear; Case No. 9, the right) died of causes other than the operation.

Case 6, a gifted young man with a University degree was operated bilaterally, the right ear with sufficient, the left with insufficient, hearing gain. During his first sojourn abroad this patient committed suicide while in a manic depressive attack from which he had been suffering some time. One week before his death he wrote of how wonderful it was to have regained the hearing in his right ear and of the new hope for the future that it gave him.

Case 9 was a debilitated boy of 10 years with bilateral congenital ear atresia and a cleft palate, who presented the following operative findings:

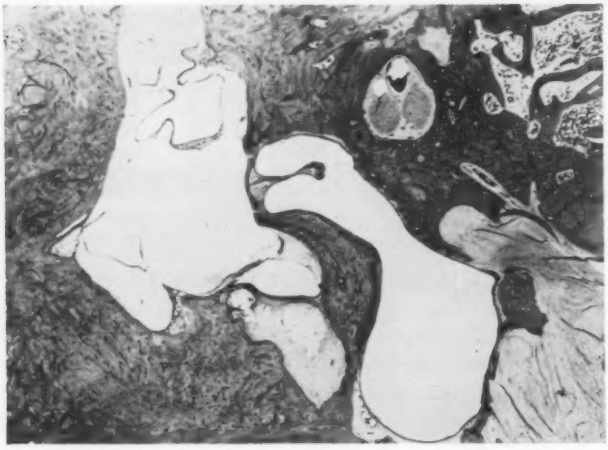

Fig. 7. Case No. 9. Male, 10 years. Atresia auris congenita. Left temporal bone (not operated). (Vertical serial section). Section through the window-niche.

Small mastoid process without pneumatization. Large ventral displacement of the sigmoid sinus. Very low middle fossa, consequently very cramped relationship. Finally a very small antrum and a small aditus were opened. Connective tissue found in both spaces was removed. An

ossicle resembling an incus could then be extracted. The stapes could not be seen. Formation of a skin flap to line the newly formed external canal and to close the middle ear in the operative cavity was done.

Post-operatively there was a clear but for practical purposes insufficient hearing gain.

Six weeks following the operation the patient sustained a fatal accident.

Histological examination of the petrous bone.

Left temporal bone (not operated) (vertical serial section). Section through the window. (See Fig. 7). In the lateral wall of the middle ear cavity, the tympanic bone, the tympanic membrane and the external meatus are missing. The resulting defect was partially closed by a downward extension of the squama temporalis. On the other side a bony lamella extends upward from the lateral part of the floor of the middle ear. The defect in the lateral wall is filled with strands of dense connective tissue. The squama is situated very close to the oval window of the inner ear capsule so that the tympanic cavity is extremely low. In this bony bridge the ventrally displaced facial nerve runs. The oval window, the foot plate of the stapes and the ligament are normal. The low tympanic cavity is so small here that the head of the stapes (see Fig. 8) touches the bony wall of the facial canal and is joined to it by

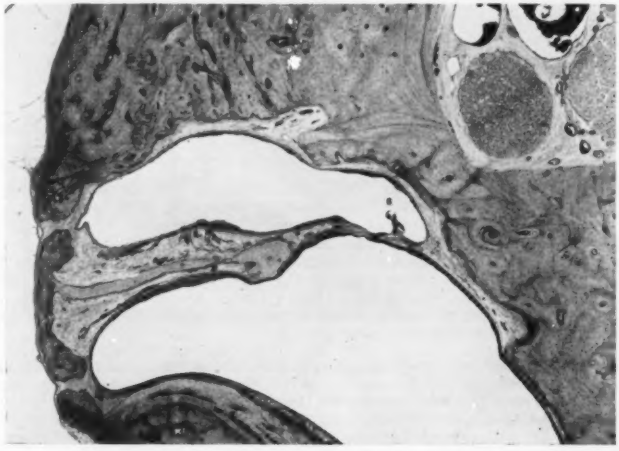

Fig. 8. Case No. 9. Male, 10 years. Atresia auris congenita. Left temporal bone (not operated). Fibrous adhesion between the capitulum stapedis and the bony wall of the facial canal.

connective tissue. Other than the stapes there are no auditory ossicles seen in this slide. The niche of the round window appears completely filled with a loose connective tissue in all sections. The inner ear is normal.

Section through the aditus ad antrum: In a fairly wide lumen a remnant of the incus lies in a wide meshwork of connective tissue, without connection to the stapes.

Right temporal bone (Pattee operation) (vertical serial section). Section through the window: (See Fig. 9). In the lateral wall of the mid-

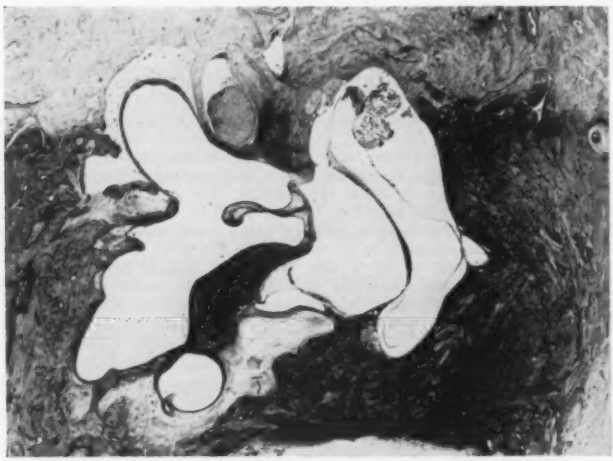

Fig. 9. Case No. 9. Male, 10 years. Atresia auris congenita. Right temporal bone (Pattee operation). (Vertical serial section). Section through the window-niche.

dle ear cavity the tympanic bone, the tympanic membrane and the external meatus are missing. The resulting defect is completely filled out by a downward extension of the squama temporalis. The middle ear cavity on this side extends way above the upper edge of the oval window to the widened tegmen tympani. The facial nerve here runs normally, in front of the upper edge of the oval window. The oval window, the foot plate of the stapes and the ligament are normal. On this side also the niche of the round window is filled completely with a loose, partially myxomatous connective tissue.

Section through the medial part of the capitulum stapedis: The capitulum is plump and broad and is bound to the bony canal of the facial nerve by a broad band of connective tissue. The narrow lumen of the tympanic cavity is lined with cuboidal epithelium whose intact surface is covered with a layer of inflammatory exudate. Extensive inflammatory infiltrations are found in the loose mucosal connective tissue.

Section through the medial part of the newly formed meatus acusticus externus: The tube of squamous epithelium terminating here shows superficial desquamation. The tunica propria shows inflammatory infiltrations.

Section through the lateral part of the newly formed meatus acusticus externus:

On the basis of similar histological changes, F. Altmann has already called attention to the fact "there may exist a fixation of the stapes by bony bridges to the medial wall or bridges between the auditory ossicles and the medial superior or outer wall which would remain undiagnosed by present methods of clinical examination."

F. Altmann and De Graaf Woodman suggest that the operative failures due to fixation of the stapes or occlusion of the niche of the round window should be fenestrated in a second phase. Since we do our otosclerosis operations in two stages routinely the same approach to the treatment of atresia seems most convenient and suitable.

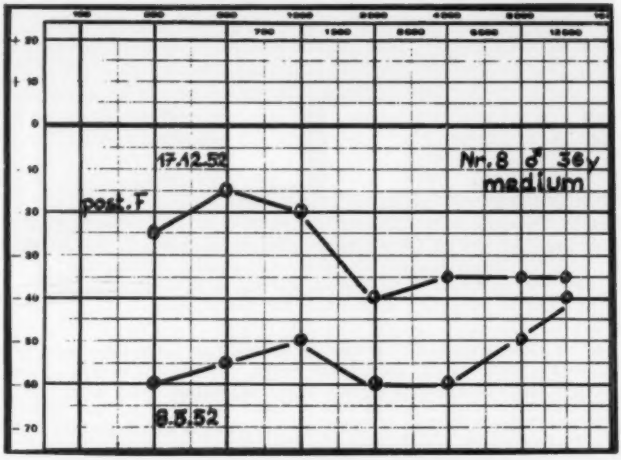

Fig. 10. Case No. 8. Audiogram of May 8, 1952, after Pattee operation. Audiogram of December 17, 1953, after secondary fenestration.

In Case No. 8 (the right ear) was fenestrated 11 months after the Pattee operation which had produced insufficient hearing gain. Two weeks post-op, the audiogram (see Fig. 10) shows a considerable improvement. Three other ears from the group of failures (Cases No. 2, 3 and 7) were suited for secondary fenestration. Two will be operated upon. The third case (Case No. 2) was meanwhile fenestrated in the second ear by Ombrédanne in a single-stage operation. Unfortunately a planned recheck of both ears could not yet be done.

Longstanding results on the operated atresia, as far as we know, are reported only by George E. Shambaugh; therefore,

we have rechecked our patients who after the Pattee operation initially showed a good to sufficient hearing gain post-operatively, with the following results: (see Fig. 11).

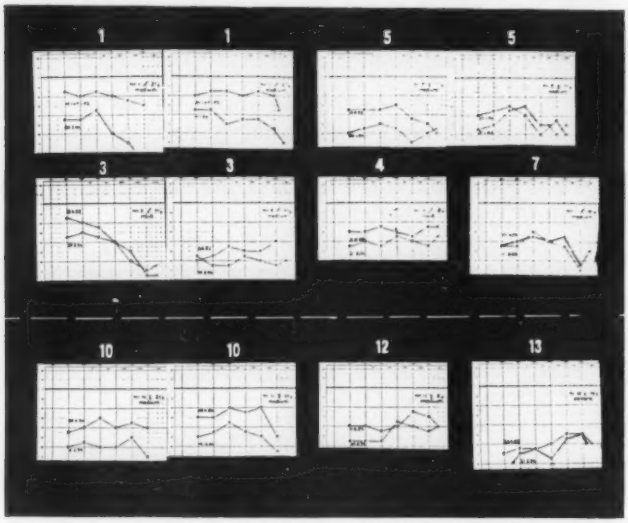

Fig. 11. Audiograms of atresia auris congenita. Longstanding results after the Pattee operation in initially successful cases.

Case No. 1 was operated by the Pattee method bilaterally with a very good hearing gain which lasted three years. Only in the course of the last two years has a hearing loss once again developed. The reason for this rather late developing hearing loss is unknown. The patient had recurrent eczematous irritation of the newly formed external meatus but was regularly treated by a specialist. In Case No. 5, operated bilaterally, the post-operative hearing gain in the right ear decreased gradually in the course of a year, while the left ear remained constant. Gradual hearing loss following an initial post-operative gain also developed in the left ear of Case No. 3, in the left ear of Case No. 4, and in three ears with combined congenital ear atresia and mandibulo-facial dysostosis.

The result of our review is anything but satisfactory. Of the 10 ears operated by the Pattee method with initially good results only two ears maintained the hearing gain after one to five years. One patient in this group died of other causes.

To arrive at some explanation for this late decline of the hearing gain we did a revision operation in Case Nos. 1, 5,

10 and 12, by lifting up the skin flap closing the middle ear. Simultaneously a fenestration of the horizontal semicircular canal was done, where possible, with the following results (see Fig. 12).

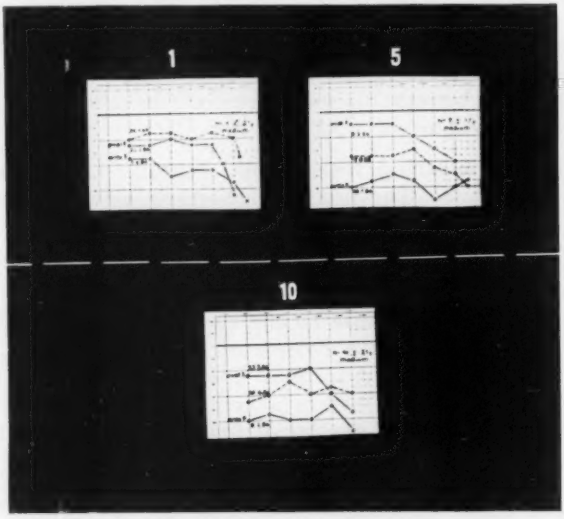

Fig. 12. Audiograms of atresia auris congenita, after revision and secondary fenestration of an initially successful Pattee operation.

In Case No. 1 (the left ear) after lifting off the skin flap the under surface showed a peculiar, gelatinous, layered arrangement of the connective tissue. In the tympanic cavity there were numerous bands of connective tissue which were removed as far as possible. Locating the horizontal semicircular canal was very difficult. A window was made in the dome and covered by a skin flap.

Re-examination and fenestration in this case resulted in a notable hearing gain. The dotted curve shows the hearing gain obtained by the Pattee operation which remained at this level for three years. The drawn out lower curve in the audiogram represents hearing before fenestration. By revision and

fenestration the previous hearing gain was restored almost to the original level.

In Case No. 5 (the right ear) granulation tissue was first removed from the posterior part of the newly formed external meatus. After lifting off the skin flap, some new bone formations over the horizontal semicircular canal were found. The tympanic cavity contained newly formed connective tissue which was removed as far as possible. A fenestration could then be done without difficulty and the fenestra was covered with a skin flap.

Ten days following fenestration, hearing was extraordinarily improved. The audiometric curve now runs above the dotted line representing hearing following the Pattee operation.

In Case No. 10 (the right ear) the revision operation was extremely difficult because of the close relationship and the abnormal position of the horizontal semicircular canal, due to the mandibulo-facial dysostosis. The narrow skin canal leading into the middle ear had to be completely removed. It was impossible to see into the small tympanic cavity, partially filled with scar tissue. The horizontal semicircular canal was found dorsally displaced after a three and one-half hours' search in bone, and finally fenestrated on its posterior surface. The window was closed by a finger-like tube of skin.

Thirteen days following fenestration this complicated atresia case also showed a hearing curve exceeding the gain achieved by the Pattee operation alone (the dotted line).

In Case No. 12 (the right ear) the anatomical relationship did not at first appear unsuitable for a revision despite the combination of atresia with dysostosis mandibulo-facialis; since here the malformation was restricted to the tympanic cavity alone (that is, the outer ear), the canal and a small immobile ear drum were present.

After lifting the skin flap, newly developed connective tissue adhesions were found and removed. In four hours of boring it was impossible to locate the horizontal semicircular canal. Finally, under the assumption that the inner ear had

been damaged, the search was abandoned. Subsequently this fear was fortunately not substantiated. Despite hours of unsuccessful drilling in the labyrinth the child hears as much following the revision, as before.

In the course of four revisions of initially successful Pattee operations, newly formed connective tissue was found in the middle ear cavity and removed. It is quite possible that this scar tissue formation is partially responsible for the gradually developing hearing loss; however, removal of the scar tissue *alone* is *not* sufficient to regain hearing as is shown in Case No. 12.

A decisive hearing gain is achieved only after fenestration of these cases. As a result of our experiences with the revisions, we are today convinced of the significance of fenestration in the treatment of atresia. We will continue to operate uncomplicated atresia cases with the Pattee method first, whereby the stapes will be located, mobilized and possibly the capitulum stapedis resected. If, however, an insufficient hearing gain is thus achieved, then a secondary fenestration will be done. In those rare cases of congenital atresia combined with skull malformation, the revision, that is secondary fenestration, according to our observations, is technically so difficult and so risky that the patient should not be subjected to the operation. We find ourselves therefore, in a paradoxical situation. Precisely the topographically most difficult cases are not suitable for the simpler two-stage method; so that the complicated atresia should be done by the more difficult one-stage operation.

Accordingly, following an unsuccessful five-stage operation on the left ear, a one-stage fenestration of the right ear was undertaken in Case No. 11.

A severely thickened mucous membrane and deformed auditory ossicle were removed as completely as possible from the extremely small tympanic cavity. The gaping petro-squamosa fissure which lay directly on the tympanic cavity's mucous lining was filled with connective tissue. The tensor tympani muscle was removed. To localize the horizontal semicircular canal the facial nerve was exposed for a short distance and

the fenestration was performed in the region of the dome. The window was closed with a pedicled flap of skin and the remaining cavity lined with a Thiersch skin transplantation.

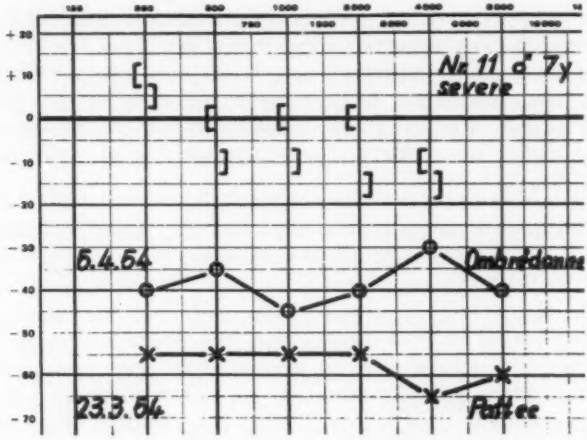

Fig. 13. Audiogram of March 23, 1954, after Pattee operation of the left ear. Audiogram of April 5, 1954, after Ombrédanne operation of the right ear.

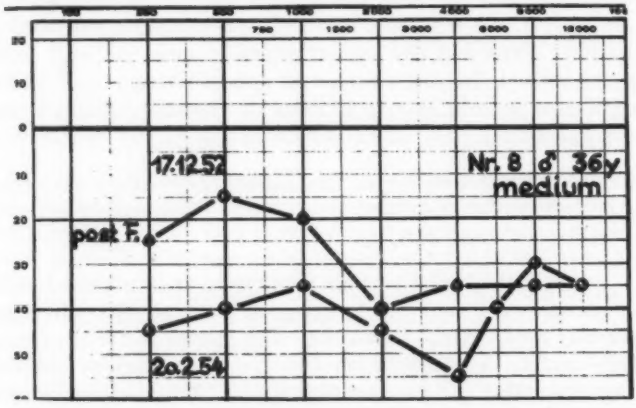

Fig. 14. Audiogram of December 17, 1952, after secondary fenestration. Audiogram of February 20, 1954, showing a hearing loss after the initially successful fenestration.

The audiometric curve (see Fig. 13) of the fenestrated right ear is closely above the Pattee operated left ear.

Finally the important question as to the permanent results of the fenestration treatment of congenital ear atresia presents itself. Unfortunately for this consideration we have only the one test patient. In this (Case No. 8) a decrease of the hearing gains due to fenestration occurred in the course of two years (see Fig. 14). Naturally we cannot draw any conclusions from one such observation. George E. Shambaugh has reported two fenestrated atresia cases with still sufficient hearing improvement three years after the operation; therefore, it seems worthwhile to continue our surgical efforts. It seems important that we also obtain further observation on fenestrated atresia cases.

SUMMARY.

A total of 19 ears in 12 cases of bilateral ear atresia and one case of one-sided atresia, were operated upon. Nine of these cases were uncomplicated atresia, four were atresia combined with mandibulo-facial dysostosis. In 10 out of 18 ears a good to adequate hearing gain was first achieved by the Pattee method. Operative failure could be histologically verified in one case as due to fixation of the stapes. In seven out of 10 Pattee cases the operatively achieved hearing gain was lost in the course of several years. By revision and fenestration the initial hearing gain could be restored or exceeded, in three cases.

For uncomplicated cases of ear atresia the two-stage fenestration operation, combined with stapes mobilization and possible resection in the first phase is recommended. Ear atresia, combined with malformations of the skull, should be fenestrated in a one-stage procedure. The prognosis of the fenestrated atresia cases has yet to be determined on the basis of the material outstanding.

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POST GRADUATE SESSION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY.

A Post Graduate Session in Ophthalmology and Otolaryngology sponsored by the Virginia Society of Ophthalmology and Otolaryngology will meet at the University of Virginia from November 30 to December 3, 1954.

November 30 and December 1, will be devoted to Otolaryngology lectures and December 2 and 3, will be devoted to Ophthalmological subjects. Those interested please contact Dr. Edwin Burton or Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Va.

PROGRESSIVE ORAL GANGRENE DUE TO ACATALASEMIA (COLORED MOTION PICTURE).*†

SHIGEO TAKAHARA, M.D.,
(By Invitation),
Chicago, Ill.

About five years ago the author encountered an eight-year-old Japanese girl with acatalasemia, *i.e.*, the absence of catalase in the blood. There was a peculiar progressive gangrene starting in the gum of the upper jaw which encroached upon the maxilla, maxillary sinus, and nasal cavity. The infected areas were broadly excised, and on application of hydrogen peroxide during lavage, the wound surface promptly changed into a brownish black color, and no foaming of the hydrogen peroxide solution occurred. Investigations were started to account for this strange phenomenon, and it became apparent that the lack of catalase in the patient's blood was responsible for the blackish discoloration of the blood by hydrogen peroxide. In normal individuals, hydrogen peroxide is decomposed by the catalase in the blood, into $2\ H_2O_2 \longrightarrow 2\ H_2O + O_2$, giving off oxygen in the form of bubbles, and the blood remains a bright red color. In contrast, if there is an absence of catalase in the blood, as in the above mentioned patient, no decomposition of the applied hydrogen peroxide results; therefore, it actively oxidizes the hemoglobin and changes it into methemoglobin, which in turn accounts for the brownish black discoloration. In *in vitro* reaction, upon leaving the reagent for about 30 minutes iron-globin precipitation results leaving a clear supernatant. This supernatant is propentdyopent, the final breakdown product of hemoglobin, which shows a positive pentdyopent reaction.

Of the six siblings in this family, four (including the above patient) had acatalasemia. It was common among these four to show a progressive gangrene originating in the teeth. We

* Presented at the 58th Annual Meeting of the American Laryngological, Rhinological, and Otological Society, Inc., Boston, Mass, May, 1954.

† From the Department of Otolaryngology, Okayama University Medical School, Okayama, Japan.

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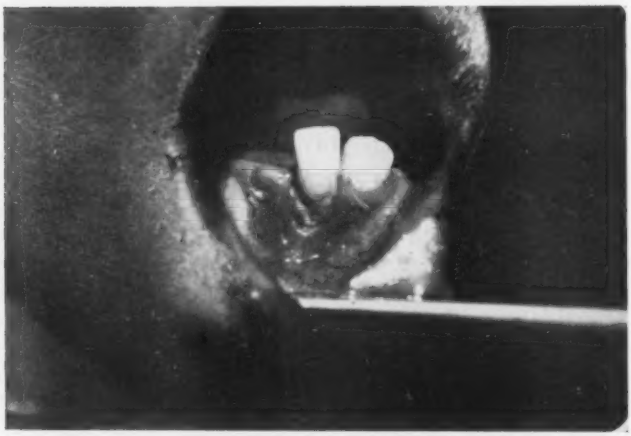
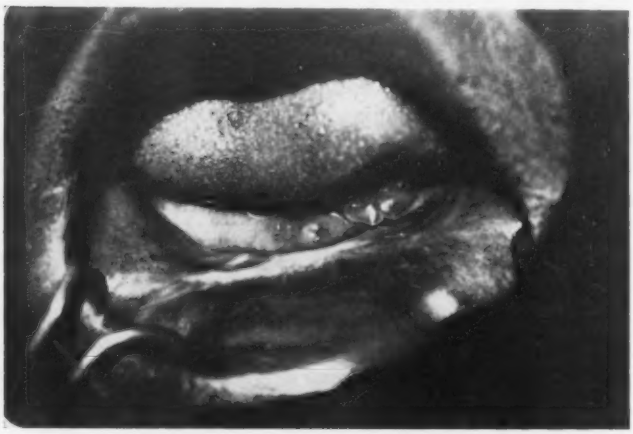


CASE 3. AGE 8, FEMALE, MODERATE TYPE.
Early stadium of ulcer starting around the root of the frontal teeth in the lower jaw.

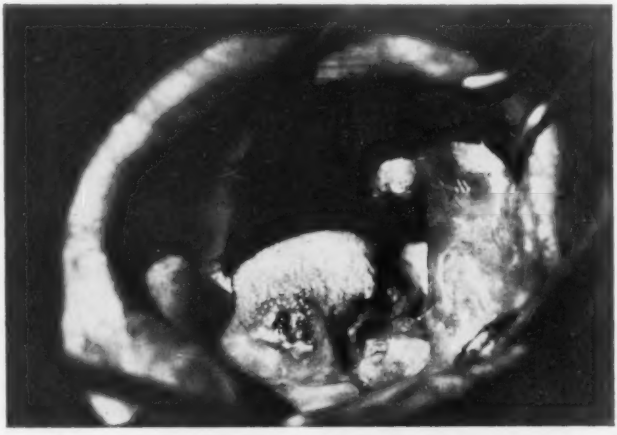


CASE 5. AGE 16, MALE, SEVERE TYPE.
Though the oral disease is now healed, there can be seen a remarkable recession of the gum. This patient cannot wear denture as the raised area of the gum is too low to be fitted.

CASE 5. AGE 16, MALE, SEVERE TYPE.

The healed condition of the upper jaw. The picture shows a remarkable recession of gum and cicatricial mucous membrane of the cheek which are characteristic of this disease.

Post-operative perforation communicates with nasal cavity and maxillary sinus.



CASE 5. AGE 16, MALE, SEVERE TYPE.

The ulcer which started around the back tooth of lower jaw, is gradually developing into the lateral surface of tongue. Also slight ulcer on the mucous membrane of cheek is noticeable. Most of the tooth had already fallen out.

suspected a relationship between the peculiar dental lesions and acatalasemia, and subsequent studies have verified this impression.

A continual vigilance, in the belief that other cases of similar character existed, was rewarded with two additional families which added five more cases to my series. Thus a total of three family lines with nine cases have now been investigated. Observations during the past four years indicated that catalase value of the blood in all nine cases were invariably zero, with no rise at all when tests were made.

Since my first report, 1947, seven corroborative cases of acatalasemia have been added to my nine cases, making a total of 16 cases in Japan. Fourteen of these 16 cases, namely over 80 per cent, showed the peculiar and characteristic oral gangrene during their childhood, from the age of two to ten. In all, the lesion was what may be termed a serious alveolar pyorrhea or a slowly progressing type of noma, and unless adequate treatment was given to check the progress it would progress and occasionally bring about serious systemic symptoms.

In the history of our nine cases, there are no records of receiving irradiation as from Atomic Bomb or X-ray treatment, and each of 16 cases occurred in several different places in Japan. This suggests that they have no tendency to be characteristic of certain areas.

The treatment consisted of extraction with thorough and extensive excision of the gangrenous bones and involved soft tissues. The results were usually good. Continual rinsing of the infected regions and administration of penicillin also proved an effective adjunct.

It should be added that consanguineous marriages were noted in the parents of all three families. This coupled with the fact that all nine cases of acatalasemia which have come under my observation were present in these families have led me to believe that hereditary factors are undoubtedly responsible or influence this unusual condition. The transmission is believed to be of the Mendelian recessive monogenetic character.

This study was presented in colored motion pictures.

A CASE OF OTITIS EXTERNA APPARENTLY CAUSED BY SALMONELLA FLORIDA.*

WILLIAM KILLINGER,†
MARGARET WYNN,†
and
FLORENCE YOUNG,‡
Jacksonville, Fla.

The literature contains reports of the isolation of Salmonella in a wide variety of clinical entities, including otitis media. We believe this is the first report of Salmonella infection in otitis externa.

The case here reported was one of more than 500 observed during the summer of 1953 in a special study of otitis externa at the U. S. Naval Air Station, Jacksonville, Florida. Cultures were taken routinely from each affected ear on admission of the patient to the clinic and three times a week during clinical follow-up. The specimen was obtained by inserting a sterile cotton swab into the ear canal, then placing the swab at once in thioglycollate broth. In mid-afternoon the cultures were transported to the laboratories of the Florida State Board of Health for examination. When the specimens arrived, ordinarily two to six hours after the culture was taken, plates of eosin methylene-blue and blood agar were inoculated by a standard loop.

The patient, a 21-year-old white enlisted man, was admitted to the clinic on July 8, 1953. He complained of pain in the right ear, unilateral difficulty in hearing, a feeling of fullness in the ear canal, and discomfort on opening the mouth and on biting. The auricle was tender on manipulation. Examination revealed mild erythema and edema of the auditory canal and a scanty gray discharge. The drum was well visualized and found to be normal. A culture was taken in the routine manner. The ear canal was irrigated with 3 per cent saline, and a wick saturated with terramycin-polymyxin B solution, was inserted. The pa-

* Studies of Otitis Externa were conducted under Contract AF 33 (038) 28643 with the USAF School of Aviation Medicine, Randolph Field, Texas, Dept. of Otolaryngology, Washington University School of Medicine, St. Louis, Missouri (B. H. Senturia, M.D., Director), and Contract AF 33 (038)-12465 with the Bureau of Laboratories, Florida State Board of Health, Jacksonville, Florida (A. V. Hardy, M.D., Director).

† Medical students at Tulane and Miami Universities respectively, serving clinic assistants during the summer.

‡ Bacteriologist, Florida State Board of Health.

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tient was also given 1 cc. of the terramycin-polymyxin B solution, in a dropper bottle with instructions to apply two drops into the ear canal every three hours. The culture was submitted for examination in the usual way.

The patient returned the following day according to clinic routine and reported he was much improved and free of discomfort. Examination revealed less erythema and edema and a small amount of exudate in the canal. Treatment was repeated as on the preceding day. He was unable to return for 15 days (July 23, 1953), at which time he reported that he had had no further trouble with his ear and had stopped using the drops at the end of the second day of treatment. His ear canal was normal and a repeat culture revealed only a non-hemolytic *staphylococcus albus*. Two fecal cultures were obtained, and these were negative for enteric pathogens.

The original ear culture, when plated routinely to eosin methylene-blue and blood agar plates, revealed virtually a pure growth of *S. florida*. There were numerous colonies indicating that the purulent exudate inoculated contained large numbers of organisms. All colonies picked gave the typical biochemical and serologic reactions of *S. florida*.* In vitro sensitivity tests indicated that the organism was very sensitive to terramycin and sulfadiazine.

This case is of interest as the first reported *Salmonella* infection of the external auditory canal. Bacteriologic observations have clearly established that Gram-negative organisms, notably Pseudomonas, are commonly encountered in this condition. Although rarely occurring in the normal ear, these organisms are found in large numbers in the infected external auditory canal.

The observation reported here indicates that a known enteric pathogen, also a Gram-negative organism, occasionally may grow luxuriantly in the ear canal and presumably may cause external otitis. The public health implication of this finding is significant, since obviously a food handler with an ear infection discharging an enteric pathogen could contaminate food and be the source of epidemic disease. The prompt clinical response suggested that the terramycin-polymyxin B solution was an effective therapeutic agent. There was no indication as to the probable source of the infecting organism.

To evaluate dependably the frequence of occurrence of Salmonella in otitis externa, more sensitive culture techniques for this organism would be required. The use of an appropriate enrichment, either tetrathionate or selenite F, in an adequate series of cases would be justified.

* The identification and typing were confirmed by Mildred Gaiton, Bacteriologist, U. S. Public Health Service, assigned to Florida.

UNUSUAL FOREIGN BODY (PORCUPINE QUILL) IN THE ESOPHAGUS. CASE REPORT.

H. D. HARLOWE, M.D.,

Miles City, Mont.

Foreign bodies of the esophagus are not infrequent or uncommon, but occasionally unusual ones are found. From a review of the literature, Hill[1] reported two cases of porcupine quills as laryngeal foreign bodies, but none have been found in the esophagus. According to Hill,[1] "The ingestion of porcupine quills is extremely rare, and it seems fortunate that they tend to implant themselves firmly in any adjacent tissue and thus remain in an easily accessible region."

Matheson,[2] in reviewing over 600 cases of foreign bodies of the esophagus, stresses the importance of an esophagoscopy even after negative X-ray in any case of suspected foreign body of the esophagus.

REPORT OF CASE.

History: The patient, P. N. E., a white female, age 87 years and edentulous, stated that six days previously she was eating hamburger and believed she swallowed a small sharp piece of bone. She consulted her physician and was told that it was probably just a scratch of her esophagus and to return in a few days if it did not improve. She returned in five days, still complaining of dysphagia and pain in the region of the cricopharyngeus. The patient was referred with a tentative diagnosis of possible foreign body of the cervical esophagus.

Treatment and Course: Preliminary X-rays revealed no definite evidence of any radiopaque foreign body of the esophagus; however, on the lateral cervical esophagus film made after the swallowing of thin barium, a narrow non-opaque foreign body showed in the cervical esophagus outlined by adhering barium at the level of the fifth cervical vertebra.

An esophagoscopy was performed, and what was assumed to be a small sliver of bone was removed with little difficulty. The patient was observed for two days following the operation and then discharged recovered.

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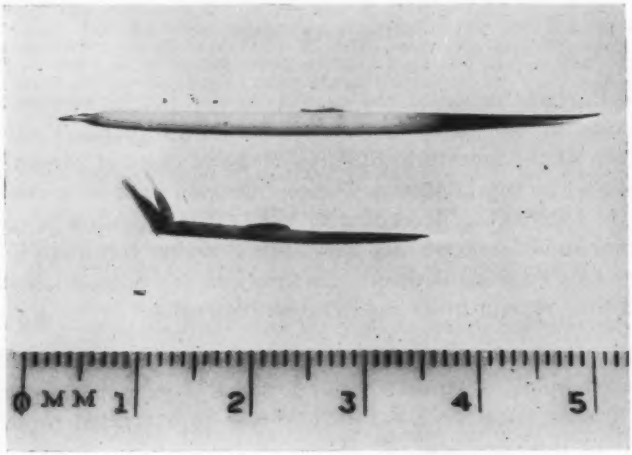
COMMENT.

The foreign body was later found to be a "porcupine quill." The hamburger the patient had eaten was made from deer and pork meat. The porcupine quill had passed through the



meat grinder and one portion of the quill shows where it was damaged. The quill measured over 25 mm. in length, and the larger one in the picture is for comparison only.

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2. MATHESON, I.: Foreign Bodies in the Esophagus, a Review of Six Hundred and Two Cases. Jour. Laryngol., and Otol., 435-445, Aug., 1949.

Garberson Clinic.

AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY, INC.

The American Otorhinologic Society for Plastic Surgery, Inc., will hold its meeting at the Waldorf-Astoria Hotel, New York City, September 19, 1954. Participants in the program will be Drs. W. A. McNichols, Sr., J. G. Gilbert, Harry Neivert, Matthew S. Ersner, John J. Conley, and John Jenney. For further information contact Dr. L. J. Feit, 66 Park Ave., New York (16), N. Y.

SUSPENSION FIXATION GEAR POWER LARYNGOSCOPY (WITH MOTION PICTURES).*

ROBERT B. LEWY, M.D.,
Chicago, Ill.

The material which I present is specifically dependent upon considerable prior history and the inventive accomplishments of others. The formulation of both the instruments and instrument combination depends upon work dating back at least 100 years. In direct laryngoscopy, reference should first be made to the work of Desmoreaux in 1853. This was followed in turn by that of Kirstein and shortly later Killian; both contributed to the direct tubular view. Important accessories to the tubular instruments were devised by Seiffert and Haslinger with the former contributing a chest support, and the latter improving somewhat on this with a gear system. Marked improvements were made with a manual distal lighted tube by Jackson.

Jackson's contributions on teaching of proper procedure were of great importance. The open field of suspension work, while started by Killian, received its greatest impetus from R. C. Lynch. For important and extensive cord or tumor surgery this is the method of choice in many clinics.

The instrument combination which I present is a lineal descendant of the laryngoscope of Seiffert and Jackson. The former modified by Haslinger, Roberts, Andrews and Somers. The instrument combination contains some of the strongest and simplest characteristics of each, I trust with the best features in one system. The position method similarly is an easy working simplification.

In my personal experience with the standard method of head holding in the "Boyce position" and with the usual tubular laryngoscope used without mechanical aid, there was often a problem. I found I was occasionally neither strong

* Presented at the 58th Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Boston, Mass., May 26, 1954.

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enough nor perhaps skillful enough to expose the anterior commissure without great difficulty. There was as well an occasional necessity for general anesthesia or its equivalent. For some years I presumed that this was a personal problem, and let it go at that. Finally, mustering up some courage, I began to inquire among my colleagues. I drew some cold comfort from the fact that many of them candidly admitted that they had frequently contended with similar situations.

A fresh approach to the problem was instituted when one of our residents at the Hines Veterans Hospital made a copy of the window casement opener laryngoscope holder that Lt. Col. Somers presented in the April, 1952, *Archives of Oto-*

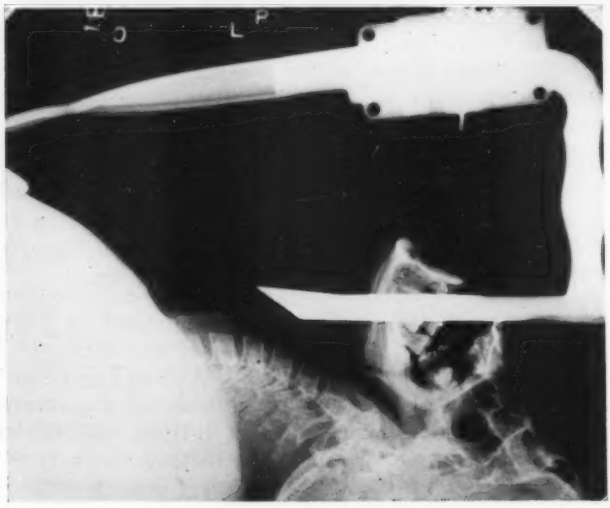

Fig. 1. X-ray Laryngoscope with Holder.

laryngology. This was a home-made imitation of the Andrews detachable laryngoscope holder; however, it possessed a striking difference: there was adequate available power (something Somers did not describe) through the gear system of the casement opener. This was a combined sprocket and worm gear.

There were large gear teeth that moved easily and rapidly. These could not slip, since they integrated a vertical gear system into an almost horizontal groove system of the upright worm.

Using this feature of the Somers instrument as the most essential factor, this power gear was made the functional center of a detachable holder. This had a strong, easily used three-point grip which grasped the laryngoscope handle.

In an independent venture a horizontal bar was put on the Lynch apparatus. With this modification the gallows and the table clamp were dispensed with, and the laryngoscope holder substituted in their place.

We have found that the gear power lifts the tip of the laryngoscope and exposes the anterior commissure gradually and easily. We have used it repeatedly, now in many of scores of cases and thus far with only two failures. Once the necessary exposure is attained, two hands and a relaxed and balanced body position are available for biopsy, removal of polyp or fibroma, papilloma or for exacting examination of the subglottic area after retracting a vocal cord.

Contrary to the orthodox teaching, we found no difficulty in performing laryngoscopy with the neck extended and the vertex of the head on the table as the motion picture will show. This position seemed of itself to hold the shoulders down, making it possible to dispense with either mechanical or human head holders, and thus to reduce by two the team membership requirements of other methods.

The next slide is an X-ray film of a patient thus positioned and with the instrument in place. The mouth and larynx are in a direct line. The neck is extended and the laryngoscope clears the cervical spine easily, as you can see.

In the motion picture that we are about to show you, we will demonstrate a non-standard position for direct laryngoscopy and an available power system attachable to standard laryngoscopes.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

August 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 700.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model "Mercury."
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T-10; Microtone Model T-612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny Myte).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor; 2 tubes. Model A-330; 3 transistors. Model A-335; 3 transistors.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.
Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32. Illinois.

Maico Transist-Ear, Model O; 3 transistors.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes.
Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarion Model D-1; 3 transistors. Otarion Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-25; 3 transistors.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.
Distributor: Sears, Roebuck & Co., 925 South Homan Ave., Chicago 7, Illinois.

Sonotone Model 1010; 1 transistor, 2 tubes. Model No. 1111; 3 transistors.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes. Telex Model 956; 3 transistors.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex Hearing Aids (three types).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

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Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Hollywood Hotel, Hollywood, Fla., March 17-18, 1955.

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Librarian-Historian: Dr. Bernard J. McMahon, St. Louis, Mo.
Meeting: Hollywood Hotel, Hollywood, Fla., March 13-14, 1955.

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President-Elect. Dr. Dean M. Lierle, Iowa City, Iowa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Hollywood Hotel, Hollywood, Fla., March 15-16-17, 1955, morning only.

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Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Waldorf-Astoria, New York City, Sept. 19-24, 1954.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Hotel Marshall, Richmond, Va., March 6-10, 1955.
Waldorf-Astoria, New York City, Sept. 13-17, 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Clyde A. Heatley, 11 No. Goodman St., Rochester, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Hollywood Beach Hotel, Hollywood, Fla., March 15-16, 1955, afternoon only.

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Secretary: Dr. Jack L. Levine.
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Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

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Meeting: Waldorf-Astoria, New York City, September, 1954.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

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Meeting: Chicago, Ill., Sept. 22-24, 1954.

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Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

Spring Meeting: Natural Bridge Hotel, Natural Bridge, Va., May 7-8, 1955.

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Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
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Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

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Meeting: Waldorf-Astoria Hotel, New York, N. Y., Sept. 19, 1954.

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Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

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Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

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Meeting: Honolulu, 1954.

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Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

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President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

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President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: U. S. A., 1957.

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CONTENTS